What is claimed is:

1. A method to identify agonist ligands of progesterone receptors, comprising:

a. contacting a progesterone receptor with a putative agonist ligand, wherein said progesterone receptor is selected from the group consisting of progesterone receptor A (PR-A) and progesterone receptor B (PR-B), under conditions wherein, in the absence of said putative agonist ligand, said progesterone receptor is not activated;

b. detecting expression of at least one gene that is regulated by said progesterone receptor when said progesterone receptor is activated, said at least one gene being selected from the group consisting of:

i. at least one gene that is selectively upregulated by PR-A chosen from a gene in Table 1;

ii. at least one gene that is selectively downregulated by PR-A chosen from a gene in Table 2;

iii. at least one gene that is selectively upregulated by PR-B chosen from a gene in Table 3;

iv. at least one gene that is selectively downregulated by PR-B chosen from a gene in Table 4;

v. at least one gene that is upregulated or downregulated by both PR-A and PR-B chosen from a gene in Table 5;

vi. at least one gene that is reciprocally regulated by PR-A and PR-B chosen from a gene in Table 6; and,

vii. at least one gene that is regulated by one of said PR-A or said PR-B, wherein regulation of said gene is altered when the other of said PR-A or PR-B is expressed by the same cell, chosen from a gene in Table 7; and,

c. comparing the expression of said at least one gene in the presence and in the absence of said putative agonist ligand, wherein detection of regulation of the expression of said at least one gene in the manner associated with activation of said progesterone receptor as set forth in (b) indicates that said putative agonist ligand is a progesterone receptor agonist.

2. The method of Claim 1, wherein said progesterone receptor is PR-A.

3. The method of Claim 1, wherein said progesterone receptor is PR-B.

4. The method of Claim 1, wherein said progesterone receptor comprises both PR-A and PR-B.

5. The method of Claim 1, wherein detection of upregulation of expression of at least one gene chosen from a gene in Table 1, or detection of downregulation of at least one gene chosen from a gene in Table 2, in the presence of said putative agonist ligand, indicates that said putative agonist ligand is a selective agonist of PR-A.

6. The method of Claim 1, wherein detection of upregulation of expression of at least one gene chosen from a gene in Table 3, or detection of downregulation of at least one gene chosen from a gene in Table 4, in the presence of said putative agonist ligand, indicates that said putative agonist ligand is a selective agonist of PR-B.

7. The method of Claim 1, wherein said step (b) of detecting comprises detecting expression of at least five genes from any one or more of said Tables 1-7.

8. The method of Claim 1, wherein said step (b) of detecting comprises detecting expression of at least ten genes from any one or more of said Tables 1-7.

9. The method of Claim 1, wherein said step (b) of detecting comprises detecting expression of at least 15 genes from any one or more of said Tables 1-7.

10. The method of Claim 1, further comprising a step of detecting expression of at least one gene chosen from the genes in Table 8.

11. The method of Claim 1, wherein said progesterone receptor is expressed by a cell.

12. The method of Claim 11, wherein said progesterone receptor is endogenously expressed by said cell.

13. The method of Claim 11, wherein said progesterone receptor is recombinantly expressed by said cell.

14. The method of Claim 11, wherein said cell is part of a tissue from a test animal.

15. The method of Claim 14, wherein said step of contacting is performed by administration of said putative agonist ligand to said test animal or to said tissue of said test animal.

16. The method of Claim 1, wherein expression of said at least one gene is detected by measuring amounts of transcripts of said at least one gene before and after contact of said progesterone receptor with said putative agonist ligand.

17. The method of Claim 1, wherein expression of said at least one gene is detected by detecting hybridization of at least a portion of said at least one gene or a transcript thereof to a nucleic acid molecule comprising a portion of said at least one gene or a transcript thereof in a nucleic acid array.

18. The method of Claim 1, wherein expression of said at least one gene is detected by measuring expression of a reporter gene that is operatively linked to at least the regulatory region of said at least one gene.

19. The method of Claim 1, wherein expression of said at least one gene is detected by detecting the production of a protein encoded by said at least one gene.

20. The method of Claim 1, wherein said putative agonist ligand is a product of rational drug design.

21. The method of Claim 1, comprising, in step (b), detecting expression of: 11-beta-hydroxysteroid dehydrogenase type 2, tissue factor gene, PCI gene (plasminogen activator inhibitor 3), MAD-3 Ikβ-alpha, Niemann-Pick C disease (NPC1), platelet-type phosphofructokinase, phenylethanolamine n-methyltransferase (PNMT), transforming growth factor-beta 3 (TGF-beta3), Monocyte Chemotactic Protein 1, delta sleep inducing peptide (related to TSC-22), and estrogen receptor-related protein (hERRa1).

22. The method of Claim 1, comprising, in step (b), detecting expression of: growth arrest-specific protein (gas6), tissue factor gene, NF-IL6-beta (C/EBPbeta), PCI gene (plasminogen activator inhibitor), Stat5A, calcium-binding protein S100P, MSX-2, lipocortin II (calpactin I), selenium-binding protein (hSBP), and bullous pemphigoid antigen (plakin family).

23. The method of Claim 1, comprising, in step (b), detecting expression of phenylethanolamine n-methyltransferase (PNMT) adrenal medulla.

24. The method of Claim 1, comprising, in step (b), detecting expression of proteasome-like subunit MECL-1.

25. The method of Claim 1, comprising, in step (b), detecting expression of: growth arrest-specific protein and tissue factor gene.

26. A method to identify antagonists of progesterone receptors, comprising:

a. contacting a progesterone receptor with a putative antagonist ligand, wherein said progesterone receptor is selected from the group consisting of progesterone receptor A (PR-A) and progesterone receptor B (PR-B), under conditions wherein, in the absence of said putative antagonist ligand, said progesterone receptor is activated;

b. detecting expression of at least one gene that is regulated by said progesterone receptor when said progesterone receptor is activated, said at least one gene being selected from the group consisting of:

i. at least one gene that is selectively upregulated by PR-A chosen from a gene in Table 1;

ii. at least one gene that is selectively downregulated by PR-A chosen from a gene in Table 2;

iii. at least one gene that is selectively upregulated by PR-B chosen from a gene in Table 3;

iv. at least one gene that is selectively downregulated by PR-B chosen from a gene in Table 4;

v. at least one gene that is upregulated or downregulated by both PR-A and PR-B chosen from a gene in Table 5;

vi. at least one gene that is reciprocally regulated by PR-A and PR-B chosen from a gene in Table 6; and,

vii. at least one gene that is regulated by one of said PR-A or said PR-B, wherein regulation of said gene is altered when the other of said PR-A or PR-B is expressed by the same cell, chosen from a gene in Table 7; and,

c. comparing the expression of said at least one gene in the presence and in the absence of said putative antagonist ligand, wherein detection of inhibition or reversal of the regulation of expression of said at least one gene as compared to the regulation of expression of said at least one gene in the manner associated with activation of said progesterone receptor as set forth in (b), indicates that said putative antagonist ligand is a progesterone receptor antagonist.

27. The method of Claim 26, wherein said progesterone receptor is PR-A.

28. The method of Claim 26, wherein said progesterone receptor is PR-B.

29. The method of Claim 26, wherein said progesterone receptor comprises both PR-A and PR-B.

30. The method of Claim 26, wherein said progesterone receptor is activated by contacting said receptor with a compound that activates said receptor, said step of contacting being performed prior to, simultaneously with, or after said step of contacting of (a).

31. The method of Claim 26, wherein detection of inhibition of expression or downregulated expression of at least one gene chosen from a gene in Table 1 in the presence of said putative antagonist ligand as compared to the expression of said at least one gene in the presence of said compound that activates said progesterone receptor, or detection of inhibition of expression or upregulation of expression of at least one gene chosen from a gene in Table 2 in the presence of said putative antagonist ligand as compared to the expression of said at least one gene in the presence of said compound that activates said progesterone receptor, indicates that said putative antagonist ligand is a selective antagonist of PR-A.

32. The method of Claim 26, wherein detection of inhibition of expression or downregulation of expression of at least one gene chosen from a gene in Table 3 in the presence of said putative antagonist ligand as compared to the expression of said at least one gene in the presence of said compound that activates said progesterone receptor, or detection of inhibition of expression or upregulation of expression of at least one gene chosen from a gene in Table 4, in the presence of said putative antagonist ligand as compared to the expression of said at least one gene in the presence of said compound that activates said progesterone receptor, indicates that said putative antagonist ligand is a selective antagonist of PR-B.

33. The method of Claim 26, wherein said step (b) of detecting comprises detecting expression of at least five genes from any one or more of said Tables 1-7.

34. The method of Claim 26, wherein said step (b) of detecting comprises detecting expression of at least ten genes from any one or more of said Tables 1-7.

35. The method of Claim 26, wherein said step (b) of detecting comprises detecting expression of at least 15 genes from any one or more of said Tables 1-7.

36. The method of Claim 26, further comprising a step of detecting expression of at least one gene chosen from the genes in Table 8.

37. The method of Claim 26, wherein said progesterone receptor is expressed by a cell.

38. The method of Claim 37, wherein said progesterone receptor is endogenously expressed by said cell.

39. The method of Claim 37, wherein said progesterone receptor is recombinantly expressed by said cell.

40. The method of Claim 37, wherein said cell is part of a tissue from a test animal.

41. The method of Claim 40, wherein said step of contacting is performed by administration of said putative agonist ligand to said test animal.

42. The method of Claim 26, wherein expression of said at least one gene is detected by measuring amounts of transcripts of said at least one gene before and after contact of said progesterone receptor with said putative agonist ligand.

43. The method of Claim 26, wherein expression of said at least one gene is detected by detecting hybridization of at least a portion of said at least one gene or a transcript thereof to a nucleic acid molecule comprising a portion of said at least one gene or a transcript thereof in a nucleic acid array.

44. The method of Claim 26, wherein expression of said at least one gene is detected by measuring expression of a reporter gene that is operatively linked to at least the regulatory region of said at least one gene.

45. The method of Claim 26, wherein expression of said at least one gene is detected by detecting the production of a protein encoded by said at least one gene.

46. The method of Claim 26, wherein said putative antagonist ligand is a product of rational drug design.

47. A method to identify isoform-specific agonists of progesterone receptors, comprising:

a. contacting a progesterone receptor with a putative agonist ligand, wherein said progesterone receptor is selected from the group consisting of progesterone receptor A (PR-A) and progesterone receptor B (PR-B), under conditions wherein in the absence of said putative agonist ligand, said progesterone receptor is not activated;

b. detecting expression of at least one gene that is regulated by said progesterone receptor when said progesterone receptor is activated, said at least one gene being selected from the group consisting of:

i. at least one gene that is exclusively upregulated or downregulated by PR-A, chosen from a Table selected from the group consisting of Table 1 and Table 2; and,

ii. at least one gene that is exclusively upregulated or downregulated by PR-B chosen from a Table selected from the group consisting of Table 3 and Table 4; and,

c. comparing the expression of said at least one gene in the presence and in the absence of said putative agonist ligand, wherein detection of regulation of the expression of said at least one gene in the manner associated with activation of said progesterone receptor as set forth in (b)(i) but not (b)(ii), indicates that said putative agonist ligand is a PR-A-specific agonist, and wherein detection of regulation of the expression of said at least one gene in the manner associated with activation of said progesterone receptor as set forth in (b)(ii) but not(b)(i), indicates that said putative agonist ligand is a PR-B-specific agonist.

48. The method of Claim 47, wherein said progesterone receptor comprises both PR-A and PR-B.

49. The method of Claim 47, wherein said step (b) of detecting comprises detecting expression of at least five genes from any one or more of said Tables 1-4.

50. The method of Claim 47, wherein said step (b) of detecting comprises detecting expression of at least ten genes from any one or more of said Tables 1-4.

51. The method of Claim 47, wherein said step (b) of detecting comprises detecting expression of at least 15 genes from any one or more of said Tables 1-4.

52. A method to identify isoform-specific antagonists of progesterone receptors, comprising:

a. contacting a progesterone receptor with a putative antagonist ligand, wherein said progesterone receptor is selected from the group consisting of progesterone receptor A (PR-A) and progesterone receptor B (PR-B), under conditions wherein, in the absence of said putative antagonist ligand, said progesterone receptor is activated;

b. detecting expression of at least one gene that is regulated by said progesterone receptor when said progesterone receptor is activated, said at least one gene being selected from the group consisting of:

i. at least one gene that is exclusively upregulated or downregulated by PR-A, chosen from a Table selected from the group consisting of Table 1 and Table 2; and,

ii. at least one gene that is exclusively upregulated or downregulated by PR-B chosen from a Table selected from the group consisting of Table 3 and Table 4; and,

c. comparing the expression of said at least one gene in the presence and in the absence of said putative antagonist ligand, wherein, in the presence of said putative antagonist ligand, detection of inhibition or reversal of the regulation of expression of said at least one gene as compared to the regulation of expression of said at least one gene in the manner associated with activation of said progesterone receptor as set forth in (b)(i) but not (b)(ii), indicates that said putative antagonist ligand is a PR-A-specific antagonist, and wherein, in the presence of said putative antagonist ligand, detection of inhibition or reversal of the regulation of expression of said at least one gene as compared to the regulation of the expression of said at least one gene in the manner associated with activation of said progesterone receptor as set forth in (b)(ii) but not (b)(i), indicates that said putative antagonist ligand is a PR-B-specific antagonist.

53. The method of Claim 52, wherein said progesterone receptor comprises both PR-A and PR-B.

54. The method of Claim 52, wherein said step (b) of detecting comprises detecting expression of at least five genes from any one or more of said Tables 1-4.

55. The method of Claim 52, wherein said step (b) of detecting comprises detecting expression of at least ten genes from any one or more of said Tables 1-4.

56. The method of Claim 52, wherein said step (b) of detecting comprises detecting expression of at least 15 genes from any one or more of said Tables 1-4.

57. A method to identify a tissue-specific agonist of a progesterone receptor, comprising:

a. providing an expression profile for at least one gene that is known to be regulated by a progesterone receptor in both a first and second tissue type when said progesterone receptor is activated, wherein said at least one gene is chosen from the genes in any one or more of the genes in Tables 1-7;

b. contacting a progesterone receptor expressed by a first tissue type with a putative agonist ligand, wherein said progesterone receptor is selected from the group consisting of progesterone receptor A (PR-A) and progesterone receptor B (PR-B), under conditions wherein, in the absence of said putative agonist ligand, said progesterone receptor is not activated;

c. contacting a progesterone receptor expressed by a second tissue type with said putative agonist ligand under conditions wherein, in the absence of said putative agonist ligand, said progesterone receptor is not activated, wherein said progesterone receptor is the same isoform as the progesterone receptor contacted in (b);

d. detecting expression of said at least one gene from (a);

e. comparing the expression of said at least one gene in the presence and in the absence of said putative agonist ligand in each of said first and second tissue types, wherein detection of regulation of the expression of said at least one gene in one of said first or second tissue types in the manner associated with activation of said progesterone receptor as set forth in said expression profile of (a), and detection of inhibition of regulation or no regulation of said at least one gene in the other of said first or second tissue types, as compared to the expression of said at least one gene associated with activation of said progesterone receptor as set forth in said expression profile of (a), indicates that said putative agonist ligand is a tissue-specific progesterone receptor agonist.

58. The method of Claim 57, wherein said first tissue type is breast, and wherein said at least one gene is selected from the group consisting of:

i. at least one gene that is selectively upregulated by PR-A chosen from a gene in Table 1;

ii. at least one gene that is selectively downregulated by PR-A chosen from a gene in Table 2;

iii. at least one gene that is selectively upregulated by PR-B chosen from a gene in Table 3;

iv. at least one gene that is selectively downregulated by PR-B chosen from a gene in Table 4;

v. at least one gene that is upregulated or downregulated by both PR-A and PR-B chosen from a gene in Table 5;

vi. at least one gene that is reciprocally regulated by PR-A and PR-B chosen from a gene in Table 6; and,

vii. at least one gene that is regulated by one of said PR-A or said PR-B, wherein regulation of said gene is altered when the other of said PR-A or PR-B is expressed by the same cell, chosen from a gene in Table 7.

59. The method of Claim 57, wherein said second tissue type is selected from the group consisting of breast, uterus, bone, cartilage, cardiovascular tissues, heart, lung, brain, meninges, pituitary, ovary, oocyte, corpus luteum, oviduct, fallopian tubes, T lymphocytes, B lymphocytes, thymocytes, salivary gland, placenta, skin, gut, pancreas, liver, testis, epididymis, bladder, urinary tract, eye, and teeth.

60. The method of Claim 57, wherein said first tissue type is a non-malignant tissue and wherein said second tissue type is a malignant tissue from the same tissue source as the first tissue type.

61. The method of Claim 60, wherein said tissue source is breast tissue.

62. The method of Claim 57, wherein said first tissue type is a normal tissue and wherein said second tissue type is a non-malignant, abnormal tissue.

63. The method of Claim 57, wherein said expression profile of genes regulated by a progesterone receptor in said first or second tissue type is provided by a method comprising:

a. providing a first cell of a selected tissue type that expresses a progesterone receptor A (PR-A) and not a progesterone receptor B (PR-B) and a second cell of the same tissue type that expresses PR-B and not PR-A;

b. stimulating said progesterone receptors in (a) by contacting said first and second cells with a progesterone receptor stimulatory ligand;

c. detecting expression of genes by said first and second cells in the presence of said stimulatory ligand and in the absence of said stimulatory ligand, wherein a difference in the expression of a gene in the presence of said stimulatory ligand as compared to in the absence of said stimulatory ligand, indicates that said gene is regulated by said progesterone receptor in said selected tissue type.

64. A method to identify a tissue-specific antagonist of a progesterone receptor, comprising:

a. providing an expression profile for at least one gene that is known to be regulated by a progesterone receptor in both a first and second tissue type when said progesterone receptor is activated, wherein said at least one gene is chosen from the genes in any one or more of the genes in Tables 1-7;

b. contacting a progesterone receptor expressed by a first tissue type with a putative antagonist ligand, wherein said progesterone receptor is selected from the group consisting of progesterone receptor A (PR-A) and progesterone receptor B (PR-B), under conditions wherein, in the absence of said putative antagonist ligand, said progesterone receptor is activated;

c. contacting a progesterone receptor expressed by a second tissue type with said putative antagonist ligand, wherein said progesterone receptor is selected from the group consisting of progesterone receptor A (PR-A) and progesterone receptor B (PR-B), under conditions wherein, in the absence of said putative antagonist ligand, said progesterone receptor is activated;

d. detecting expression of said at least one gene from (a); and,

e. comparing the expression of said at least one gene in the presence and in the absence of said putative antagonist ligand in each of said first and second tissue types, wherein detection of regulation of the expression of said at least one gene in one of said first or second tissue types in the manner associated with activation of said progesterone receptor as set forth in said expression profile of (a) in the presence of said putative antagonist ligand, and detection of inhibition or reversal of regulation of expression of said at least one gene in the other of said first or second tissue types in the presence of said putative antagonist ligand, as compared to the expression of said at least one gene associated with activation of said progesterone receptor as set forth in said expression profile of (a), indicates that said putative antagonist ligand is a tissue-specific progesterone receptor antagonist.

65. The method of Claim 64, wherein said first tissue type is breast, and wherein said at least one gene is selected from the group consisting of:

i. at least one gene that is selectively upregulated by PR-A chosen from a gene in Table 1;

ii. at least one gene that is selectively downregulated by PR-A chosen from a gene in Table 2;

iii. at least one gene that is selectively upregulated by PR-B chosen from a gene in Table 3;

iv. at least one gene that is selectively downregulated by PR-B chosen from a gene in Table 4;

v. at least one gene that is upregulated or downregulated by both PR-A and PR-B chosen from a gene in Table 5;

vi. at least one gene that is reciprocally regulated by PR-A and PR-B chosen from a gene in Table 6; and,

vii. at least one gene that is regulated by one of said PR-A or said PR-B, wherein regulation of said gene is altered when the other of said PR-A or PR-B is expressed by the same cell, chosen from a gene in Table 7.

66. The method of Claim 64, wherein said second tissue type is selected from the group consisting of breast, uterus, bone, cartilage, cardiovascular tissues, heart, lung, brain, meninges, pituitary, ovary, oocyte, corpus luteum, oviduct, fallopian tubes, T lymphocytes, B lymphocytes, thymocytes, salivary gland, placenta, skin, gut, pancreas, liver, testis, epididymis, bladder, urinary tract, eye, and teeth.

67. The method of Claim 64, wherein said first tissue type is a non-malignant tissue and wherein said second tissue type is a malignant tissue from the same tissue source as the first tissue type.

68. The method of Claim 67, wherein said tissue source is breast tissue.

69. A method to identify a tissue-specific agonist of a progesterone receptor, comprising:

a. providing an expression profile for at least one gene that is known to be regulated by a progesterone receptor in a first tissue type but not a second tissue type when said progesterone receptor is activated, wherein said at least one gene is chosen from the genes in any one or more of the genes in Tables 1-7;

b. contacting a progesterone receptor expressed by said first tissue type with a putative agonist ligand, wherein said progesterone receptor is selected from the group consisting of progesterone receptor A (PR-A) and progesterone receptor B (PR-B), under conditions wherein, in the absence of said putative agonist ligand, said progesterone receptor is not activated;

c. detecting expression of said at least one gene from (a);

d. comparing the expression of said at least one gene in the presence and in the absence of said putative agonist ligand in said first tissue type, wherein detection of regulation of the expression of said at least one gene in said first tissue type in the manner associated with activation of said progesterone receptor as set forth in said expression profile of (a) indicates that said putative agonist ligand is a tissue-specific progesterone receptor agonist for said first tissue type.

70. A method to identify a tissue-specific antagonist of a progesterone receptor, comprising:

a. providing an expression profile for at least one gene that is known to be regulated by a progesterone receptor in a first but not in a second tissue type when said progesterone receptor is activated, wherein said at least one gene is chosen from the genes in any one or more of the genes in Tables 1-7;

b. contacting a progesterone receptor expressed by a first tissue type with a putative antagonist ligand, wherein said progesterone receptor is selected from the group consisting of progesterone receptor A (PR-A) and progesterone receptor B (PR-B), under conditions wherein, in the absence of said putative antagonist ligand, said progesterone receptor is activated;

c. detecting expression of said at least one gene from (a); and,

d. comparing the expression of said at least one gene in the presence and in the absence of said putative antagonist ligand in said first tissue type, wherein detection of inhibition or reversal of regulation of expression of said at least one gene in said first tissue type in the presence of said putative antagonist ligand, as compared to the expression of said at least one gene associated with activation of said progesterone receptor as set forth in said expression profile of (a), indicates that said putative antagonist ligand is a tissue-specific progesterone receptor antagonist of said first tissue type.

71. A method to determine the profile of genes regulated by progesterone receptors in a breast tumor sample, comprising:

a. obtaining from a patient a breast tumor sample;

b. detecting expression of at least one gene in said breast tumor sample that is regulated by a progesterone receptor when said progesterone receptor is activated, said at least one gene being selected from the group consisting of:

i. at least one gene that is selectively upregulated by PR-A chosen from a gene in Table 9;

ii. at least one gene that is selectively downregulated by PR-A chosen from a gene in Table 10;

iii. at least one gene that is selectively upregulated by PR-B chosen from a gene in Table 11;

iv. at least one gene that is selectively downregulated by PR-B chosen from a gene in Table 12;

v. at least one gene that is upregulated or downregulated by both PR-A and PR-B chosen from a gene in Table 13;

vi. at least one gene that is reciprocally regulated by PR-A and PR-B chosen from a gene in Table 14; and,

vii. at least one gene that is regulated by one of said PR-A or said PR-B, wherein regulation of said gene is altered when the other of said PR-A or PR-B is expressed by the same cell, chosen from a gene in Table 15; and,

c. producing a profile of genes for said tumor sample that are regulated selectively by PR-A, selectively by PR-B, or by both PR-A and PR-B.

72. A plurality of polynucleotides for the detection of the expression of genes regulated by progesterone receptors in breast tissue;

wherein said plurality of polynucleotides consists of polynucleotide probes that are complementary to RNA transcripts, or nucleotides derived therefrom, of genes that are regulated by progesterone receptors; and

wherein said plurality of polynucleotides comprises polynucleotide probes that are complementary to RNA transcripts, or nucleotides derived therefrom, of genes selected from the group consisting of:

a. at least one gene that is selectively upregulated by PR-A chosen from a gene in Table 1;

b. at least one gene that is selectively downregulated by PR-A chosen from a gene in Table 2;

c. at least one gene that is selectively upregulated by PR-B chosen from a gene in Table 3;

d. at least one gene that is selectively downregulated by PR-B chosen from a gene in Table 4;

e. at least one gene that is upregulated or downregulated by both PR-A and PR-B chosen from a gene in Table 5;

f. at least one gene that is reciprocally regulated by PR-A and PR-B chosen from a gene in Table 6; and,

g. at least one gene that is regulated by one of said PR-A or said PR-B, wherein regulation of said gene is altered when the other of said PR-A or PR-B is expressed by the same cell, chosen from a gene in Table 7.

73. The plurality of polynucleotides of Claim 72, wherein said polynucleotide probes are immobilized on a substrate.

74. The plurality of polynucleotides of Claim 72, wherein said polynucleotide probes are hybridizable array elements in a microarray.

75. The plurality of polynucleotides of Claim 72, wherein said polynucleotide probes are conjugated to detectable markers.

76. The plurality of polynucleotides of Claim 72, wherein said plurality of polynucleotides further comprises at least one polynucleotide probe that is complementary to RNA transcripts, or nucleotides derived therefrom, of at least one gene chosen from the genes in Table 8.

77. A plurality of antibodies, or antigen binding fragments thereof, for the detection of the expression of genes regulated by progesterone receptors in breast tissue;

wherein said plurality of antibodies, or antigen binding fragments thereof, consists of antibodies, or antigen binding fragments thereof, that selectively bind to proteins encoded by genes that are regulated by progesterone receptors; and

wherein said plurality of antibodies, or antigen binding fragments thereof, comprises antibodies, or antigen binding fragments thereof, that selectively bind to proteins encoded by genes selected from the group consisting of:

a. genes that are selectively upregulated by PR-A chosen from genes in Table 1;

b. genes that are selectively downregulated by PR-A chosen from genes in Table 2;

c. genes that are selectively upregulated by PR-B chosen from genes in Table 3;

d. genes that are selectively downregulated by PR-B chosen from genes in Table 4;

e. genes that are upregulated or downregulated by both PR-A and PR-B chosen from genes in Table 5;

f. genes that are reciprocally regulated by PR-A and PR-B chosen from genes in Table 6; and,

g. genes that are regulated by one of said PR-A or said PR-B, wherein regulation of said gene is altered when the other of said PR-A or PR-B is expressed by the same cell, chosen from genes in Table 7.

78. The plurality of antibodies, or antigen binding fragments thereof, of Claim 77, wherein said plurality of antibodies, or antigen binding fragments thereof, further comprises at least one antibody, or an antigen binding fragment thereof, that selectively binds to a protein encoded by a gene chosen from the genes in Table 8.

79. A method to identify genes that are regulated by a progesterone receptor in two or more tissue types, comprising:

a. activating a progesterone receptor in two or more tissue types that express said progesterone receptor;

b. detecting expression of at least one gene said two or more tissue types, said at least one gene being chosen from a gene in any one or more of Tables 1-7, and,

c. identifying genes that are regulated by said progesterone receptor in each of said two or more tissue types.

80. The method of Claim 79, further comprising detecting whether said genes are regulated selectively by PR-A, selectively by PR-B, or by both PR-A and PR-B.

81. A method to regulate the expression of a gene selected from the group consisting of any one or more of said genes in Tables 1-7, wherein said method comprises administering to a cell that expresses a progesterone receptor a compound selected from the group consisting of: progesterone, a progestin, and an antiprogestin, wherein said compound is effective to regulate the expression of said gene.

82. The method of Claim 81, wherein said gene is selected from the group consisting of: growth arrest-specific protein (gas6), NF-IL6-beta (C/EBPbeta), calcium-binding protein S100P, MSX-2, selenium-binding protein (hSBP), and bullous pemphigoid antigen (plakin family).

83. The method of Claim 81, wherein said cell that expresses a progesterone receptor is in the breast tissue of a patient that has, or is at risk of developing, breast cancer.